



**SCHOOL OF PSYCHOLOGY – DOCTORATE IN CLINICAL PSYCHOLOGY**  
**MAJOR RESEARCH PROJECT**

**Preventing and Predicting Oppositional Defiant Disorder**

**Submitted by Rebecca Phillips, to the University of Exeter,**  
**as a thesis for the degree of Doctorate of Clinical Psychology, May 2016**

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**SCHOOL OF PSYCHOLOGY – DOCTORATE IN CLINICAL PSYCHOLOGY**

**MAJOR RESEARCH PROJECT**

**Literature Review: A systematic review of classroom based preventative interventions  
for oppositional defiant disorder.**

Trainee Name: **Rebecca Phillips**

Primary Research Supervisor: **Dr Heather O'Mahen**

University of Exeter

Secondary Research Supervisor: **Dr Anke Karl**

University of Exeter

External Supervisor: **Prof Dale Hay**

Cardiff University

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### **Abstract**

*Background:* Oppositional Defiant Disorder (ODD) is a relatively common childhood disorder that is associated with a number of adverse outcomes. It is often identifiable from a young age, with younger onset predicting more adverse outcomes. Preventative interventions have therefore been developed that aim to reduce the onset of ODD and its symptoms. A number of school-based preventative interventions have been implemented, but no review of their effectiveness has been conducted.

*Objectives:* To conduct a systematic review of classroom based preventative interventions for oppositional defiant disorder.

*Method:* Electronic databases in the psychological and educational fields were systematically searched for evidence of school-based preventative interventions.

*Results:* Eight studies were identified that met the inclusion and exclusion criteria. These ranged from large universal based studies, to selected and indicated types of prevention. There were methodological flaws in a number of studies, particularly with regards to random assignment. Therefore, the conclusions drawn from these studies are tentative.

*Conclusions:* There was some evidence for the effectiveness of school-based interventions for ODD. Further research is needed, with higher quality designs, to replicate and extend the findings to date.

**KEYWORDS:** Oppositional Defiant Disorder; Prevention; School-based.

## Introduction

Oppositional defiant disorder (ODD) describes a pattern of argumentativeness, hostility, and noncompliance towards authority figures. It is a disorder that is typically present before the age of eight (Kazdin, 1995) and has an average age of onset of six years (Hinshaw & Anderson, 1996). Problems of aggression and oppositional behaviour associated with ODD are a frequent psychopathology in children and adolescents (Hunter, 2003), and are the most common reasons why young children are referred to mental health services in the UK (NICE, 2014). Child ODD places strain on public services, such as educational settings, and is associated with future social, emotional and behavioural difficulties (Burke, Waldman, & Lahey, 2010).

Childhood behavior problems tend to be stable over time, and the stability is greater the earlier the problems manifest (Robins, 1991). Children with early onset behavioral difficulties are at particular risk for later disruptive behaviour (Loeber, Burke, & Pardini, 2009), and for internalizing problems such as depression (Boylan, Vail-lancourt, & Szatmari, 2012). There is also evidence from longitudinal analyses that ODD is associated with an “early starter” pathway to conduct problems, (Patterson, Capaldi, & Bank, 1991). Indeed, the developmental cascades model posits that interacting systems affect development in a cumulative and interactive fashion, with varying environmental components impacting upon the children across their development (Dodge, Greenberg, Malone, & CPPRG, 2008).

Thus, early prevention is considered the most effective type of intervention for the life-long problems that are associated with children’s emotional disorders (Hester & Kaiser, 1998). One such way of intervening is by targeting factors known to be associated with the onset of a disorder. According to the Committee on Prevention of Mental Disorders, interventions are considered to address primary prevention when they occur before the initial onset of a clinically diagnosable disorder (Munoz, Mrasek, & Haggerty, 1996). They further

divide prevention into three categories: universal, selective, and indicated. Universal programs are those that target everyone, selective when directed towards those with biological, psychological and social risk factors associated with the disorder, and indicated when the targeted individuals exhibit some of the symptoms of the disorder.

The Conduct Problems Prevention Research Group (1992) suggests that prevention should begin at the point that children can be identified as high-risk, and that this identification can begin in schools. Children with emotional and behavioural difficulties cause disruption in classrooms, and can be met with frustrated teachers (Stormshak, Bierman, McMahon, & Laengua, 2000). Furthermore, teachers report higher levels of stress with teaching students with ODD in mainstream schools (McClean and Dixon, 2010), and these difficulties will likely impact on teachers' capacity to provide for the broader classroom. NICE quality standards for the treatment of Conduct Disorders (CDs under which ODD is subsumed) suggests, as its first quality standard, that early intervention classroom-based emotional learning and problem-solving programs should be available to children at social risk for the development of CDs. A number of school-based programs are available, but to date no review has compared the effectiveness of these interventions, nor their impact on the development of ODD as a distinct diagnosis.

The classification and choice of measurement is an important consideration when comparing studies for their effectiveness. The category of disruptive behaviour disorder (DBD) contains ODD along with CD and attention deficit/hyperactivity disorder (ADHD). Often, models of child psychopathology group DBDs together. However, there are known distinctions in their aetiology and presentation (Burke, Lober, & Lahey, 2003). Therefore, studies and reviews that combine the DBDs may mask or miss specific processes related to each condition. Furthermore, there have been fewer studies that consider ODD specifically than those that have considered CD or ADHD alone.



Of particular concern to the conceptualization of ODD, as compared with other externalizing diagnoses, is that the behaviours that comprise a diagnosis of ODD are observable to some degree in the vast majority of children during their development. Young children may behave in aggressive or oppositional ways when trying to control their environment, particularly before they have developed the communicative repertoire to express their needs in less disruptive ways (Tremblay, 2000). Therefore, a diagnosis of ODD is made when the frequency, intensity and duration of these behaviours exceeds the level expected developmentally. This taxonomic issue is reflected in the varying methods for the measurement of ODD in studies of intervention. Some assume a continuous scaling, thereby employing questionnaires that measure conduct or behaviour problems generally. Others assess for the presence of ODD as a categorical and diagnostic construct. For the purposes of this review, only those studies that employ measures of clinically significant symptoms will be included, rather than focussing on the prevention of normative levels of transient oppositional behaviour that are known to decline with age (Bongers, Koot, van Der Ende, & Verhulst, 2004). Therefore, diagnostic symptom counts and full diagnostic criteria will be considered as sufficient outcome measures.

Previous systematic literature reviews have considered the effectiveness of particular treatments for ODD, although not necessarily preventative in nature. These include parent management training (Michelson, Davenport, Dretzke, Barlow, & Day, 2013), group based family interventions (Furlong, McGalloway, Bywater, Hutchings, Smith, & Donnelly 2013) and other psychosocial interventions (Dretzke, Davenport, Frew, Barlow, Stewart-Brown, Bayliss, et al., 2009). In addition, a number of reviews have considered ODD and CD together, despite the evidence that both are distinct diagnoses (Loney & Lima, 2003) that likely require varying treatments. Thus, the aim of the current investigation is to conduct a

systematic literature review regarding the effectiveness of *preventative* classroom-based interventions for ODD.

## Method

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed throughout each stage of this review (Moher, Liberati, Tetzlaff, Altman, & The PRISMA Group, 2009).

### Eligibility Criteria

**Types of studies.** Only studies that included a control group were included, and ideally those with random assignment at either an individual or school level. Quasi-experimental designs were also considered. Only those studies published in peer-reviewed journals were included.

**Types of participants.** Studies were only included if the participants were children (that is, below the age of nineteen) at the point of intervention.

**Types of interventions.** Only interventions deemed to be preventative were included. Preventative interventions are those which occur before the initial onset of a clinically diagnosable disorder; any intervention after that point is defined as treatment (Munoz, Mrasek, & Haggerty, 1996).

**Types of outcome measures.** Only studies that measured ODD or symptoms thereof as the primary outcome variable were included. Studies that measured ODD symptoms at baseline only (in order to screen) but measured other outcomes (such as reading ability) were not included. In order to investigate the effectiveness of interventions for the onset of ODD specifically, only studies that used outcome measures that matched or were significantly related to the diagnostic criteria for ODD were included.

**Exclusion criteria.** Papers were excluded if they were not available in the English language.

### **Information Sources**

The following electronic databases were searched: Australian Education Index, EBSCO (including British Education Index, EBSCO e-journals, Education Research Complete, and Psychological and Behavioural Sciences Collection), OVID (including MEDLINE: PubMed, PsychINFO, PsychARTICLES, and Embase), JSTOR, Applied Social Science Index and Abstracts, Cochrane Library, Elsevier (including Science Direct and SCOPUS), and Web of Science. Each database was searched from its beginning point through to March 2016.

### **Search**

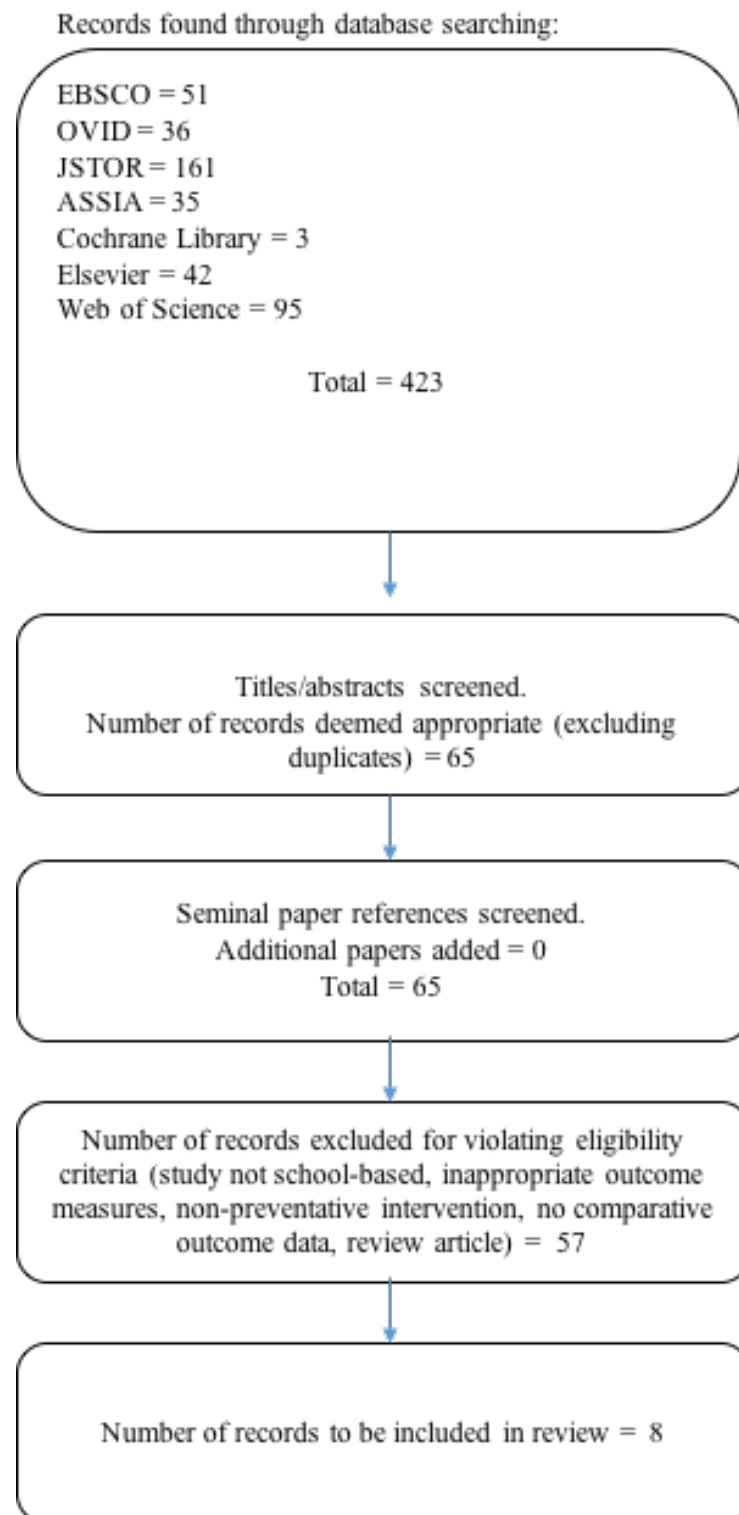
Titles and abstracts in all databases were searched to identify relevant records using the search terms: “oppositional defiant disorder” OR “oppositional defiance” AND intervention OR prevent\* OR treatment AND school OR class OR class-room OR classroom OR teacher AND random OR allocat\* OR RCT OR experiment\*.

### **Study Selection**

Titles and abstracts were initially screened to assess if they met the inclusion and exclusion criteria. Articles deemed to be potentially relevant were read in full and assessed against the eligibility criteria. Where the suitability of the study was not possible to assess via the abstract, the full text was obtained. Figure 1 displays the steps taken in selecting studies.

### **Data Extraction**

Data were extracted using the population, intervention, control and outcomes method (PICO). Key data are summarised in Table 1. Study quality was assessed using the quality assessment tool for quantitative studies (National Collaborating Centre for Methods and Tools; 2008), an objective measure of key components of quality such as study design, blinding, and selection bias.



*Figure 1.* Flow chart detailing the search strategy and process of identification, screening, eligibility and inclusion for the review.

Table 1. Studies Included in the Systematic Literature Review.

Study	N	Participants & Design	Intervention	Baseline & final follow-up point	Main outcome	Outcome measure	Study quality
Bernat, August, Hektner, & Bloomquist (2007)	T1 = 245 T6 = 151	Low-middle SES families from 26 schools. Random assignment of schools to control versus intervention conditions.	Selected and indicated. The 'Early Risers' preventative 5-year intervention.	T1 = 6 yrs & annually until T6 = 12yrs	Program children showed fewer ODD symptoms than control children at outcome. (Cohen's $d$ = .34 for parent rated ODD, .47 for youth rated ODD). Mediated by social skills and effective discipline.	NIMH DISC-IV-P and NIMH DISC-IV-Y. Parent and youth semi-structured interview regarding DSM-IV ODD symptoms and diagnosis.	Overall = strong. High drop-out rate. Reliable outcome rating and strong study design.
Bierman, et al. (2007)	891	Socially disadvantaged sample. Random assignment by school to matched intervention versus control conditions.	Selected and indicated. 'Fast Track' intervention.	T1 = 6.5 yrs Age 14 at follow-up	No main effect of the intervention on ODD, but an intervention*risk interaction; children with the highest risk showed less ODD at grade 3 (OR = 0.61).	NIMH-DISC-IV-P.	Overall = strong. Strong study design and outcome measurement.
Conduct Problems Prevention Research Group (2011)	891	Socially disadvantaged sample. Random assignment by school to matched intervention versus control conditions.	Selected and indicated. 'Fast Track' intervention.	T1 = 6.5 yrs Age 17 at follow-up	No significant main effect on ODD outcome. A marginally significant interaction effect between intervention and severity of initial risk. Significant effect of intervention on ODD outcome in those of initial high risk only (OR = 0.87).	NIMH-DISC-IV-P.	Overall = strong. Strong study design and outcome measurement. Relatively low attrition.
Froelich, Breuer, Doepfner, & Amonn (2012)	47	Within-subjects control group design with a non-randomised control group and a normal comparison group.	Indicated. A 12-week teacher training program for ADHD and ODD.	T1 = 8yrs. 6 weeks baseline wait list, and FU at end of 12 week intervention.	No effects could be identified for ODD when comparing the intervention and comparison group.	ODD Symptom Checklist (Doepfner & Lehmkuhl, 2000)	Overall = weak. Unmatched intervention vs. comparison groups, and unblinded assessment at outcome.

Study	N	Participants & design	Intervention	Baseline & final follow-up point	Main outcome	Outcome measure	Study quality
Hektner, August, Bloomquist, Lee, & Klimes-Dougan (2014)	245	Socially disadvantaged sample. Random assignment by school to matched intervention versus control conditions.	Selected and indicated. The 'Early Risers' preventative 5-year intervention.	TI = 6 yrs FU = 16 years	Program participants had fewer symptoms of ODD than controls (coefficient for total effect = $-.04$ ). This was mediated by the program's effect on social skills and parental discipline at grade 3 (coefficient for direct effect = $-.29$ ).	NIMH DISC-IV (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000)	Overall = strong. High withdrawal rate and low participation rate. Strong study design.
Peters, Petrunka, & Arnold (2003)	554	Socioeconomic disadvantage. Non-random assignment to control versus intervention conditions. Control conditions were demographically matched schools.	Universal and selected. 'Better beginnings, better futures': a community-based prevention approach. Three community sites with differing interventions.	T1 = 4 yrs FU = 8 yrs	There was a beneficial effect in oppositional symptoms following the two school-based interventions (Cohen's $d = .22$ and $.58$ ).	The oppositional behaviors subscale of the Revised Ontario Child Health Study (Boyle et al., 1993).	Overall = moderate. Weak blinding, and participant withdrawals not described. A very representative sample at baseline.
Plueck, Eichelberger, Hautmann, Hanisch, Jaenen & Doepfner (2015)	T1 = 144 FU = 42	Within-subject control group design.	Selected. Prevention Program for Externalizing Problem Behavior (PEP), 3 months.	TI = 3-6yrs FU = 4-7yrs (12-months later)	A significant reduction in ODD symptoms in the treatment group (Cohen's $d = .67$ ).	The Symptom Checklist for Disruptive Behavior Disorder (SCL-DBD) ODD scale (Doepfner et al., 2008a).	Overall = moderate. Very high drop-out rate. Strong reliability and validity of measures. Cohort design.

Study	N	Participants & design	Intervention	Baseline & final follow-up point	Main outcome	Outcome measure	Study quality
Van Lier, Muthen, van der Sar, & Crijnen (2004)	669	Randomised (at classroom level) controlled trial	Universal. The 'Good Behaviour Game' (GBG) classroom intervention	T1 = 6yrs FU = 7.5 & 8 yrs	Children were analysed for their trajectory of behavior (high, intermediate, low). The intervention had a positive effect on ODD in those in the intermediate group only (Cohen's $d = .41$ ).	Problem Behavior at School Interview (PBSI; Erasmus Medical Centre, 2007): ODD Problems Scale	Overall = strong. Strong participant selection and low withdrawal rate. Teachers provide intervention and outcome measure (via an interview with a trained researcher).

*Note.* ADHD = Attention Deficit Hyperactivity Disorder; FU = follow-up; NIMH DISC-IV-Y = National Institute of Mental Health Diagnostic Interview Schedule for Children Version IV (child interview); NIMH DISC-IV-P = National Institute of Mental Health Diagnostic Interview Schedule for Children Version IV (parent interview); ODD = Oppositional Defiant Disorder; OR = odds ratio; T1= time 1.



## Results

### Study Selection

The numbers of records accessed at each level of screening is reported in Figure 1. A random ten percent of the 65 considered studies were assessed by an independent assessor. The assessor and the author agreed in 100% of cases.

### Individual Studies

In this section, the nature of the interventions and key methodological comparisons are described, followed by a description of their results.

**Interventions.** Although each study investigated the effects of a school-based intervention on ODD, the nature of the interventions in each of the studies varied considerably. Froelich, Breuer, Doepfner, & Amonn (2012) presented a psychiatrist-led teacher training intervention, comprised of 12 sessions of education and case supervision regarding ODD and associated difficulties. Likewise, Plueck, Eichelberger, Hautmann, Hanisch, Jaenen, & Doepfner, (2015) presented a group training programme for teachers (Prevention Program for Externalizing Problem Behavior; PEP-TE). This was designed to provide teachers with behaviour modification techniques to help the child to manage emotions and build effective relationships with them in the classroom; each teacher identified one child to focus upon throughout the course of the intervention.

The ‘Good Behaviour Game’ preventive intervention (van Lier, Muthen, van der Sar, & Crijnen, 2004) was designed to promote prosocial behaviour through a consistent reward based system, and to encourage disruptive and non-disruptive peers to work together. This was a universal approach for all children in the classroom.

In addition, two long term and multi-faceted intervention programs were selected, both of which had published outcomes across multiple follow-up points. The first, ‘Early

Risers', is a multi-component intervention that involves the school and parents (Bernat, August, Hektner, & Bloomquist, 2007; Hektner, August, Bloomquist, Lee, & Kilmes-Dougan, 2014). Three components were fixed and therefore offered to all at-risk youth; a summer program, child group, and family skills parent group. A further two intervention components were made available to children on an individually-prescribed basis, and included a monitoring and mentoring school support program, along with a family support program. The complete program was implemented in two phases, a three-year intensive phase, and a further two years of less intensive booster sessions. The second, the 'Fast Track' intervention, was a ten-year program that included interventions related to the classroom environment, home-school relations, and academic skills (Bierman, Coie, Dodge, Foster, Greenberg, Lochman et al., 2007; Conduct Problems Prevention Research Group, 2011). It provided a universal classroom curriculum that aimed to promote social and emotional competence and a less aggressive social ecology within the school setting. In addition, it aimed to target poor behaviour management, poor peer relations, and poor parental monitoring and supervision.

Finally, Peters, Petrunka, and Arnold (2003) presented three different interventions in three communities respectively, two of which were considered to be school-based and so are described here. Both were considered to be universal approaches, with the aim to prevent a range of emotional and behavioural problems and promote general development. Thus, there was less specific focus on the problem of oppositional defiance or general disruptive behaviour in these interventions than in the other studies selected in this review. The Cornwall project included full-time school facilitators who provided classroom enrichment, homework help and summer tutoring as its pivotal intervention. Similarly, the Highfield project provided classroom enrichment, together with a social skills program delivered by classroom teachers.

**Study populations.** The children included in the study populations ranged from 3 to 8 years of age at the first point of the intervention, with a median age of 6. The samples were selected using staged screening criteria to identify those children at high-risk of developing conduct problems. For example, in the Fast Track study, high-risk schools were identified based on their communities' crime and poverty rates. Children were then screened by teacher ratings of the children's authority acceptance by the Teacher Observation of Child Adjustment-Revised (TOCA-R; Werthamer-Larrson, Kellman, & Wheeler, 1991), and parent rating of child behaviour using the Child Behavior Checklist (CBCL; Achenbach, 1991). Similarly, the sample of children in the Early Risers study were selected by CBCL screening of 1840 children by teachers at 20 schools. In contrast, the GBG was implemented on a universal sample, yet children were later grouped (for the purposes of analysis) according to the trajectories of disruptive behaviour (low, intermediate, and high). Therefore, the studies ranged from universal to indicated preventative interventions.

Total sample sizes at the point of intervention ranged from 47 (Froelich et al. 2012) to 891 (Bierman et al., 2007). Of the four studies whereby an intervention and control group were compared with a between-subjects randomised design, three presented groups with no significant differences in any demographic or baseline data ('Early Risers', 'Fast Track' and 'GBG'). In the two 'better beginnings, better futures' projects (Peters, Petrunka, & Arnold, 2003), the population-based interventions were evaluated by comparing the communities with other culturally and socioeconomically equivalent locations. No difference tests appear to have been calculated comparing the two; however, various risk factors were measured and included as covariates in subsequent analyses. Froelich et al. (2012) described a 'varying willingness' of teachers to take part in the teacher training intervention program, creating a difficulty in randomised or matched-pairs allocation. The teachers who took part in the training did so because they were more willing than those who did not, therefore the

motivation and qualities of the teachers may differ between the intervention and comparison groups. Furthermore, the levels of identified problem behavior differed between the intervention and control groups; the intervention group had higher baseline levels of ODD symptomology. The randomization procedure in this study is not well described.

**Outcome Measures.** Ideal assessment would include a diagnostic measure rated by more than one informant. The outcome measures varied between studies, as did the type and number of informants. The ‘Early Risers’ project was evaluated by comparing the frequency and diagnosis of ODD between its intervention and control group. ODD was measured using semi-structured psychiatric interview (NIMH DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), of both the child and one parent independently. The ‘Fast Track’ study employed the same outcome measure, but the interview was conducted with the parent only.

Froelich’s teacher training program was evaluated using the ODD symptom checklist (Doepfner & Lehmkuhl, 2000), and was completed by each child’s teacher. The same measure was employed to evaluate the PEP-TE intervention (Plueck et al., 2015), again completed by the child’s teacher. This measure assesses the diagnostic criteria for ODD according to DSM-IV and ICD-10, and has been shown to be psychometrically valid in a separate sample (Doepfner et al., 2008).

Peters, Petrunka and Arnold (2003) evaluated their two school-based interventions using the oppositional behaviours subscale of the Revised Ontario Child Health Study scale (Boyle, Offord, Racine, Fleming, Szatmari, & Sanford, 1993). Although this measure did not correspond directly to the diagnostic symptoms of ODD as the measures described above, the study remained in this review as the measure has shown adequate psychometric properties and corresponds to the ODD symptoms in a discriminant fashion (Boyle et al, 1993). Both teachers and parents completed this measure at the ages of 6, 7 and 8. Similarly, the GBG was evaluated using the Problem Behavior at School Interview (PBSI; Erasmus Medical

Centre, 2000). Trained researchers interviewed teachers about behavior that was in line with the DSM-IV criteria for ODD symptoms.

**Study Design and Quality.** The ideal randomized-control trial approach was evident in the ‘Early Risers’, ‘Fast Track’ and ‘GBG’ studies, whereby participants were allocated randomly to the intervention and control groups at the school level, and the two groups were equivalent at baseline. Peters, Petrunka, and Arnold (2003) adopted a quasi-experimental approach; a randomized control design was not possible due to the process adopted by the government in selecting the participating communities. Comparison groups were made in two ways. First a baseline-focal design, whereby the interventions participants were compared with a group of children at the same community measured at the same age some years prior. Second, a longitudinal comparison model, where changes over time in the outcome measures for children in the intervention were compared with a group of children in a comparison community, over the same time-points. Both Plueck et al. (2012) and Froelich et al. (2015) evaluated their teacher training interventions using a wait-list control design, where the change in symptoms across- and post-intervention was compared with the change in symptoms measured in a 3-month and 6-week baseline period respectively.

Studies that included multiple follow-ups were deemed to measure outcomes in a more stringent fashion. The ‘Fast Track’ intervention had a number of measurement points during the ten-year intervention, and a follow-up two years following its completion when the children were 17 (CPPRG, 2011). The Early Risers children were assessed at 12 and 16 years, 1 year and 5 years post-intervention respectively (Bernat, et al., 2007; Hektner, et al., 2014). In the remaining studies, length to follow-up varied from immediately after the end of the intervention (Froelich, et al., 2012; Peters et al., 2003), to a year post-intervention (Plueck et al., 2015).

Key to the quality of the study is the manner in which the baseline and follow-up assessments are completed. In the teacher-training studies, teachers both delivered the intervention and reported on the child's symptoms, rendering blind assessment impossible. In the two community interventions described by Peters, teachers and parents reported on the child's symptoms, preventing blind assessment. Although trained researchers conducted the interviews regarding ODD following the 'GBG', the informants were teachers, and therefore not independent. In contrast, as independent assessors were employed for both the 'Early Risers' and 'Fast Track' studies and interviewed parents/children, and the assessors were unknowledgeable about the allocation of the child, blind assessment was more likely to have occurred. This reduced the potential bias in outcome scores in these two particular studies in comparison to the previous examples.

**Results.** Main effects of intervention on ODD symptoms were found following the 'Early Risers' intervention, in the two school-based interventions presented by Peters et al. (2003), and the PEP-TE intervention. The frequency of ODD symptoms for the children in the intervention conditions was less than in those children who had experienced education-as-usual. This remained the case when the initial differences in demographic quality between the groups in the two school-based interventions were added as covariates (Peters, et al., 2003). Although no main effect of intervention was found following the 'Fast Track' intervention, there was a significant interaction between the initial risk and parent-measured ODD symptoms; those children who were particularly high-risk for developing ODD showed significantly fewer ODD symptoms at follow-up than their control group counterparts. This pattern was true for both later follow-ups at 14 and 17 years of age. The GBG showed a significant benefit in terms of ODD symptoms but only in the intermediate group of children; children with high levels of problem behavior were unaffected when compared with the control condition. No effect of the teacher training intervention described by Froelich et al.,

(2012) on ODD symptoms was found. The authors reported a decrease in symptoms across the intervention window that is different to that during the waitlist; however, the confidence intervals reported appear to discount any significance in this finding.

## **Discussion**

### **Summary of Evidence**

The current review included those studies that aimed to prevent the onset of ODD in school-aged children, in a classroom context. Studies that evaluate the effectiveness of classroom interventions in terms of preventing ODD as defined by diagnostic criteria or symptom counts are not common, with many considering the impact on normative disruptive behavior. Overall, there is evidence that classroom based interventions are able to reduce the frequency of ODD diagnosis and symptoms in childhood.

However, the effectiveness of such interventions is relatively small, with the majority of the studies included in this review showing small effect sizes. The papers with the highest ratings of study quality likely offer the most accurate measure of effectiveness, and therefore should be given the most weight. These show similarly small effect sizes. In addition, the high quality studies demonstrate that interventions do not impact every child in similar ways. While the impact of any intervention on children with low levels of baseline difficulties may be redundant due to ceiling effects, van Lier et al., (2004) found their intervention was only beneficial with children with intermediate levels of difficulties. It was not effective for those with high levels. This would appear to support an argument that those children with high levels of problem behaviour or risk are less receptive to intervention for difficulties in conduct. However, in the Fast Track intervention, it was only those children with the highest risk who were affected by the intervention positively (lower risk children were unaffected).

Therefore, it may be the case that different interventions are suitable for different types of children. While most of the children that comprised the samples included in this review were higher risk, not all the studies examined difference in treatment effects according to initial risk. Future studies may find differential effects based on these analyses, but require the statistical power and therefore the sample size to do so.

Child self-reports have been shown to be less reliable than the reports of parents or teachers (Smith, Pelham, Gnagy, Molina, & Evans, 2000), therefore it was a strength that no study relied on child reports as a primary outcome. However, a number of studies used only the teacher's rating of the child's behavior as an indicator of treatment effectiveness, although they were not independent from the intervention itself. This introduces a potential bias in the data, particularly if the teachers felt pressure to perform well, or to show that their efforts had been productive. Similarly, relying on the behavior of children in school alone also poses the risk of limiting the understanding of the effectiveness of the interventions, or indeed inflating it. For a diagnosis of ODD, problems should be present across domains (i.e. at home and at school). Therefore, using multiple informants of child outcomes would provide the most accurate estimate of intervention effectiveness.

### **Limitations**

Few studies were able to meet the criteria for this review. Those that were included were not all of high quality in terms of study design and experimental rigour, particularly in terms of level of randomisation and assessment. Conclusions about the potential for classroom preventative interventions are therefore tentatively made; particularly when regarding that the outcomes are measured by those giving the intervention itself (in the case of teachers).

The small sample of available studies did not permit any analysis of predictors, moderators or mediators of treatment effectiveness across studies. Future randomised studies



that measure relevant mediating variables, particularly those that are targeted in the intervention (such as emotion competence) should be conducted. For example, the authors of the 'Early Risers' study demonstrate that social skills and effective discipline mediate the treatment effects of their intervention. Further analysis of the potential confounding variables that may explain the effects of the intervention could be accounted for, such as general attendance at sessions, or the completion of homework, indicating whether the child receives the intended 'dose' of the intervention. Indeed, those that do and do not engage with the intervention may differ in ways that are related to the outcome of interest.

### **Recommendations**

Further evidence for the effectiveness of classroom-based prevention is required, with particular attention paid to higher quality research designs. The delineation between universal, selective and indicated prevention techniques should be addressed, including an analysis of which children would benefit from what level of intervention; particularly as the interventions presented here had differential effects depending on the level of problem behaviours. It would be illuminating to consider whether further risk factors, such as family discord or parental diagnoses, are related to the effectiveness of interventions. Furthermore, the components of the intervention should be investigated, perhaps with smaller samples, in order to build effective interventions that can then be tested in larger, more costly, studies.

### **Conclusions**

In conclusion, there are a few studies conducted to date that detail the effectiveness of preventative class-based interventions for the development of ODD specifically. Those few that are detailed in this systematic review show that a beneficial effect upon ODD symptoms is possible following classroom interventions. However, the specific effects of the classroom upon these findings is yet to be determined; two higher quality papers in this review detailed multi-component interventions, and the strictly classroom based interventions were less

experimentally rigorous. This review highlights that more evidence for the potential effectiveness of classroom preventative interventions is required. This will allow for a better understanding of the mechanisms and benefits of this hopeful approach to reducing ODD and its associated difficulties.

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**SCHOOL OF PSYCHOLOGY – DOCTORATE IN CLINICAL PSYCHOLOGY**  
**MAJOR RESEARCH PROJECT**

**Empirical Paper: Maternal depression, anger, and antisocial history as predictors of child ODD in a longitudinal community sample.**

Trainee Name: **Rebecca Phillips**

Primary Research Supervisor: **Dr Heather O'Mahen**

University of Exeter

Secondary Research Supervisor: **Dr Anke Karl**

University of Exeter

External Supervisor: **Prof. Dale Hay**

Cardiff University

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## Abstract

*Background:* Oppositional Defiant Disorder (ODD) describes a pattern of negative and disruptive behaviours in childhood that cause concurrent difficulties and are associated with later behaviour problems through to adulthood. A number of antecedents for ODD have been posited in the literature. This study explored how maternal depression, an internalising disorder, may be associated with ODD, an externalizing disorder, through its association with maternal anger. The relevance of mothers' antisocial history was also explored.

*Methods:* A community sample of 332 families were recruited to the Cardiff Child Development Study, a longitudinal design following families from pregnancy to age 7. Structural equation modeling was adopted to test a measurement model of maternal anger, then a series of hypotheses regarding the prediction of ODD when the children were 7 years of age were conducted. Maternal depression (past, antenatal and postnatal) and child ODD was measured using diagnostic clinical interview, and maternal anger through self- and partner-reported and experimenter-observation.

*Results:* Perinatal depression showed no direct relationship with ODD in this demographically diverse sample, although mothers' depression prior to pregnancy did. Maternal anger was related to ODD in families with social adversity (measured by low education, young parenthood, and unstable parental relationships). Mothers' antisocial history was a unique predictor of ODD, above that of mothers' prior depression.

*Conclusions:* Mothers' antisocial history is an important predictor of child ODD, and is also related to maternal anger. Maternal anger is related to child ODD in high adversity families. Recommendations for future research are provided.

*Keywords:* *Oppositional Defiant Disorder; Maternal Depression; Maternal Antisocial History; Maternal Anger.*

## **Introduction**

Oppositional Defiant Disorder (ODD) is the diagnostic term that describes children with a pattern of negativistic, defiant, disobedient, and hostile behaviour towards authority figures, together with marked temper tantrums and irritability. These criteria are each distinguishable from normal childhood behavior in frequency or persistence, and present a significant impact on the child's functioning at home, with peers, and/or at school. Typically, ODD is diagnosed before the age of eight (APA, 2000). ODD is one of the leading reasons for referral to child mental health services (Lober, Burke, Lahey, Winters, & Zera, 2000), and ODD is associated with later behavior and emotional problems through to adulthood (Nock, Kazdin, Hiripi & Kessler, 2007).

Whilst ODD is known to have associations with later diagnoses of Conduct Disorder (CD; Burke, Loeber, & Birmaher, 2002; Lahey et al., 1995), the majority of children with ODD do not develop CD (Lahey & Loeber, 1994). Indeed, CD specifies a more severe pattern of behaviour in which the basic rights of others or societal norms are violated. ODD is associated with increased risk for other disorders, even when accounting for its association with CD (Burke, Loeber, Lahey, & Rathouz, 2005; Harpold et al., 2007). Despite this distinction, ODD and CD are often subsumed into one category in studies of etiology. However, it is argued that the two diagnostic categories are sufficiently distinct to be studied separately, and ODD is currently under-researched in comparison. Therefore, ODD is considered as a distinct disorder here.

## **Predicting ODD**

Given the impact that ODD has concurrently, and its association with difficulties in adulthood, investigations into the potential antecedents to its onset are important for developing both preventative and acute treatments. Research has shown that disruptive

behaviour disorders (DBDs) are more prevalent in boys (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004) and there may be gender differences in the processes by which varying factors convey risk of DBDs to the developing child (Rutter, Caspi, & Moffitt, 2003). Well-documented risk factors for the development of anti-social behaviour (ASB) and DBDs include socioeconomic adversity (Arseneault, Kim-Cohen, Taylor, Caspi, & Moffitt, 2005), maladaptive parental behaviour such as substance abuse (Marmorstein, Iacono, & McGue, 2012), and family instability (Collishaw, Goodman, Pickles, & Maughan, 2007). Further recent research has aimed to explore the mechanisms by which these risk factors may convey deleterious effects on child disruptive behavior. However, less is known about the etiology of ODD independent of CD.

The goal of this proposed study is to investigate the contribution of maternal anger, measured in the postpartum period, as a mediator in the relationship between maternal depression (an internalizing disorder) and the development of ODD (an externalizing disorder) in young children, while accounting for other related risk factors. Each antecedent of interest is explored below.

### **Maternal Depression**

Maternal depression has been consistently associated with the onset of externalizing problems in children when measured in both pregnancy (e.g. Hay, Pawlby, Waters, Perra, & Sharp, 2010; Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005; O'Donnell, Glover, Barker, & O'Connor, 2014; Pemberton et al., 2010) and the postnatal period (Kim-Cohen et al., 2005). Depression is often an episodic and recurrent disorder; a history of depression is a significant predictor of perinatal depression, and women with perinatal depression are at greater risk of experiencing subsequent episodes of depression, both within future pregnancies and postnatally, and outside of the perinatal period. However, despite the chronic nature of depression, there is some evidence that the timing of depressive episodes may have

a differential effect on child outcome. Postpartum depression appears to convey a unique risk for deficits in cognitive development (Hay, Pawlby, Sharp, Asten, Mills, & Kumar, 2001; Hay et al., 2008), and depression across the perinatal period is important in the prediction of offspring emotional development (Hay, et al., 2008; Waters, Hay, Simmons, & van Goozen, 2014).

However, the evidence to date on children's emotional development is based on dimensional measures of problems, none of the studies have used standardized diagnostic interviews to determine clinically significant externalizing mental health problems in children (Waters et al., 2014). In addition, few have measured maternal depression by clinical interview; the majority have used a self-reported questionnaire-based continuous measure of maternal depression such as the Edinburgh Postnatal Depression Scale (EPDS; Cox, Holden & Sagovsky, 1987) or the Centre for Epidemiological Studies Depression Inventory (CES-D; Radloff, 1977). Relying on the same informant for both the predictor and outcome variables risks the inflation of effect sizes due to shared method variance. Furthermore, significant associations between maternal depression and externalizing problems in the child may be related to attribution biases of the informant, rather than an objective association between the two concepts.

### **Maternal Anger**

It is hypothesized that maternal anger will be positively related with child ODD, and diagnosis that has been characterized by anger. In one theory of emotional development, Eisenberg and colleagues (1998) suggest that one of the fundamental ways in which propensities to emotions develop in children is through observational learning, or through their parents' expression of emotion in the home. Studies show that anger, a component of conflict, may play a significant role in the association between marital conflict and child problems; overt and intense displays of anger and aggression by parents show more

association with child psychopathology than ratings of either covert tension in the relationship (Jenkins & Smith, 1991) or a lack of conflict resolution (Davies, Myers, & Cummings, 1995). In the current study, the mediating role of maternal anger within maternal depression is considered; one such way in which maternal depression may manifest and convey risk for externalizing disorders in children, such as ODD, is through maternal anger. Higher levels of anger in individuals with depression compared to those who are well have been consistently shown (Goldman & Haaga, 1995; Riley, Treiber, & Woods, 1989; Robbins & Tanck, 1997; Thomas, 1991; Wenzel, Gunthert, Forand, & Laurenceau, 2009). One such theory for the common heightened frequency between depression and anger is that they are both considered to be responses to the blocking of a positive reinforcer (Rolls, 1999); depression/sadness is the passive response resulting from helplessness to gain the reinforcer, and anger is a motivating response that rallies an individual to gain the reinforcer, when such action is possible. Indeed, Freud suggested that depression was ‘anger turned inward’ (1917). It may be the case, however, that individuals move between the two emotions, meeting criteria for depression whilst also experiencing anger alongside (Beck, 1996). Indeed, anger has been associated with the common, yet under-recognised, symptom of irritability in major depression (Pasquini, Picardi, Biondi, Gaetano, & Morosini, 2004).

The co-occurrence of depression and anger continues in the parental context. A mixed-method study found that women with high anger describe more negative experiences with the entry to parenthood compared to women with low anger (Parfitt & Ayers, 2012). Depression is thought to manifest in the mother-infant relationship as high levels of intrusive and/or withdrawn behaviours (Cohn, Matias, Tronick, Connell, & Lyons-Ruth, 1986), with the former style associated with anger and irritation directed at the infant (Szabo et al., 2008). Parental behaviour characterized by anger and aggression has been associated with increased oppositional behaviour in children (Dodge, Bates, & Pettit, 1990). Together, these findings

suggest that maternal anger expression may be an important avenue for investigation, both independently and in the context of depression; does maternal anger expression explain the relationship between maternal depression and child externalising difficulties?

In furthering the understanding of the prediction of ODD, along with other psychopathology, particular attention should be paid to the preschool years. Infants are affected by the emotional climate of the home from a young age, and respond differentially to anger and other emotions from very early in life. At five-months of age, infants possess the capacity to distinguish between negative emotions, even in unfamiliar languages (Fernald, 1993), and at three-months they show varied responses to anger and other emotions in their mothers' voices (Haviland & Lelwica, 1987). With this in mind, the proposed study considers the effect of maternal anger from early infancy, a stage where maternal emotion is likely to be influencing the development of the child's emotional tendencies and later child behaviour. Infants are sensitive to the emotional climate of their environment as emotions are a crucial communicative tool at this age; infants use parents' emotions to guide their behaviour in the short term (Feinmann, 1992), and their reactions to their parents' emotions predict their later socio-emotional development (Eisenberg, Cumberland, & Spinrad, 1998). Therefore, the expression of anger in maternal behaviour may impact upon the development of the child's own emotional repertoire through social learning, or by shaping their likelihood to adopt particular emotional communicative styles. Indeed, excessive anger at two years of age predicts increased risk of psychopathology at age 5 (Radke-Yarrow & Kochanska, 1990), and children with early onset disruptive behaviour problems are more likely to show poorer outcomes later in life than those who develop symptoms during adolescence (Brame, Nagin, & Tremblay, 2001; Loeber & Stouthamer-Loeber, 1998).

### **Maternal Antisocial History**



Mothers' antisocial history is associated with their children's conduct problems (Frick & Loney, 2002). Antisocial problems are also associated with depression, both generally (Romano et al., 2006; Zochillo, Meyers, & Assiter, 1997) and in the perinatal period (Cassidy, Zoccolillo, & Hughes, 1996; Hay, Pawlby, Waters, Perra & Sharp, 2010). Indeed, mothers with depression and co-occurring antisocial behavior provide poorer caregiving than mothers with depression alone (Kim-Cohen, Caspi, Rutter, Tomas, & Moffitt, 2006). It is also the case that women who experience anger are more likely to have a history of ASB; maternal hostility (defined as anger, criticism, negativity and disapproval) is related to maternal ASB (Sellers et al., 2014). Furthermore, including mothers' ASB in analyses of the relationship between maternal hostility and depression severity and child disruptive behaviour attenuates the strength of the association (Sellers et al., 2014). Therefore, it is important to consider whether it is depressed women's antisocial history that drives the relationship between depression and/or maternal anger on ODD, and/or whether a mothers' antisocial history is related to child ODD directly. It may also be the case that a mother's already established propensity to antisocial behaviour explains the relationship between maternal anger and depression in the perinatal period with later child difficulties.

### **Social Adversity**

There is also a need to consider the complex interplay between psychological and social risk factors for childhood ODD; it may be the case that, in a demographically diverse population, social adversity moderates the relationship between the hypothesised predictors and ODD. Rates of well-known risk factors, such as maternal depression, vary across population groups. Depressed mothers are more likely to draw from socioeconomically disadvantaged backgrounds and have lower educational attainment (Pickett & Wilkinson, 2010; Kim-Cohen et al., 2005), and teen pregnancy is a well-known risk factor for perinatal depression (Kim-Cohen et al., 2005). Whilst high-risk samples allow for the specific study of

mechanisms within different social groups, few studies have considered the etiology of ODD across the full range of social advantage and disadvantage. Therefore, the current study will consider whether social adversity is related to ODD directly, and/or moderates the relationship between maternal anger and ODD; it may be that maternal anger is associated with child ODD differentially, according to whether families are of particular levels of adversity.

### **Child Gender**

Externalizing difficulties are more commonly found in boys than in girls, and a greater majority of research on the development on DBDs has drawn conclusions from samples of boys. When looking exclusively at ODD, this difference in prevalence has been found to be smaller in the case of ODD than for the more severe CD (Loeber, Burke, Lahey, Winters, & Zera, 2000). Indeed, one epidemiological study has found no statistically significant difference in prevalence between genders (Nock, Kazdin, Hiripi & Kissler, 2007) and another reports that gender differences vary by the nature of the reporter (Maughan, Rowe, Messer, Goodman & Meltzer, 2004). In addition to these apparent, albeit inconsistent, differences in prevalence, differences in the etiology of DBDs have been suggested between genders. Therefore, gender will be considered as potential moderator in this data. A number of studies point to the differential susceptibility to maternal behavior between male and female infants (Martin, 1981) preschoolers (Pianta & Cardwell, 1990) and children (Webster-Stratton, 1996). For example, Martin (1981) found that non-contingent maternal caregiving was related to later infant noncompliance in boys only. Differences in the patterns of change in aggressive behaviour have been found between girls and boys as a function of parental coercion (McFayden-Ketchum, Bates, Dodge, & Petit, 1996), and overall trajectories across childhood to young adulthood are thought to differ between genders (Fontaine, Carbonneau,

Vitaro, Barker & Tremblay, 2009). Therefore, consideration for the differential effects of maternal anger on the prediction of ODD between genders may also prove important.

### **Aims and Hypotheses**

In summary, the proposed research aims to investigate a novel potential mechanism by which maternal depression, an internalizing disorder, is associated with the development of child ODD, an externalizing disorder (Pathways labelled a in Figure 1), via their potential shared association with expressed maternal anger (Pathway(s) b & c in Figure 1). The important clinical covariate of maternal antisocial history will also be included (Pathways labelled d in Figure 1), to test whether women's antisocial history has an additional effect on child ODD beyond that of the initial hypothesised relationship. As previous literature has suggested, we will test whether social adversity moderates the relationship between the clinical predictors and child ODD (Pathways labelled e in Figure 1). Finally, I will determine whether the relationships between clinical predictors and child ODD differs according to child gender (Pathways labelled f in Figure 1). These hypotheses are tested using a community sample of first-born children and their families, representative of the UK population.

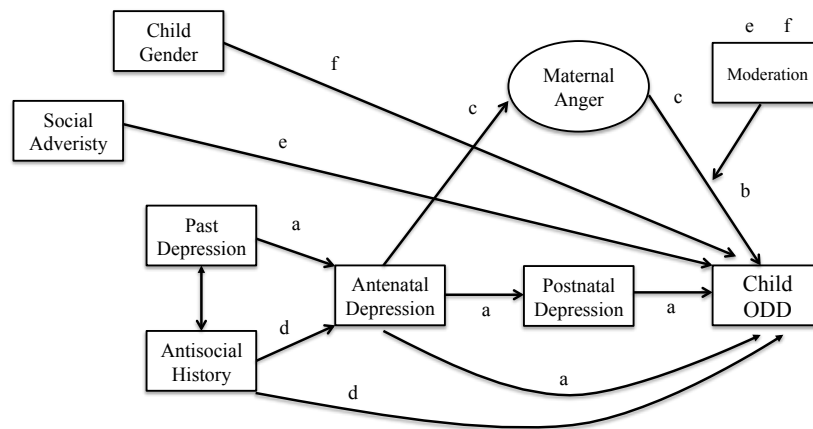


Figure 1. Hypothesised pathways.

## Method

### Design

The CCDS is a longitudinal project investigating the early prediction of violence in infancy and early childhood, with specific focus on the biological, cognitive and social risk factors for children's emotional and behavioural problems. All families entered the CCDS as expectant first-time parents. The CCDS follows families from pregnancy to the child's seventh birthday, over six time points as detailed in Table 1. The project was funded by the Medical Research Council. All procedures have been approved by the Cardiff University School of Psychology Research Ethics Committee, the NHS Multi-Centre Research Ethics Committee, and have received Research & Development approval in Wales. The current study was approved by the University of Exeter ethical approval committee, study number 2015/948.

Table 1.

*CCDS Waves and The Measures Included in the Current Study.*

CCDS Wave	Age of Child	Time Point	Measures
1	(pregnancy)	T1	Maternal depression (past and antenatal) Antisocial history Adversity
2	6 months	T2	Maternal depression (postnatal) Maternal anger
3	12 months	-	
4	18 months	-	
5	3 years	-	
6	7 years	T3	Oppositional Defiant Disorder

**Participants**

Three-hundred and thirty-two primiparous women were recruited between 1<sup>st</sup> November 2005 to 31<sup>st</sup> July 2007 from antenatal clinics in hospitals and general practice clinics in South Wales, UK. To increase the representativeness of the sample, midwifery teams also allowed researcher access to antenatal clinics for specialist medical problems, and to outreach services for vulnerably housed individuals. The majority of the recruitment took place within hospitals as GP surgeries tended to serve multiparous women rather than first-time parents.

The total recruited CCDS sample at Wave 1 (T1) was representative of the UK population in demographic qualities. The mean mother's age at birth was 28.15, 90.3% were in stable partnerships, 50.3% were married, 92.7% classed themselves as either British or Irish nationality, 59% were classified as middle class, and 79.3% of mothers had at least the basic education qualifications. Each of the recruited families gave birth to live children.

Of the original sample, 310 (94%) families were seen again following childbirth; 11 (3%) asked to leave the study, 8 (2%) were not traced within the time window, and 3 (0.9%) could not be assessed due to health reasons or adverse family circumstances. The sample at

wave 2, referred to as time 2 (T2) for the purpose of the current project, did not differ from the original sample on any demographic variable. Infants averaged an age of 6.6 months ( $SD = 0.9$ ) at the point of assessment.

At wave 6, the final wave in the CCDS in its current form, families were revisited when their child was approximately seven years of age. This wave point is classed as T3 here. A total of 268 families participated in the sixth wave in full (including a diagnostic interview rather than just a postal questionnaire).

### **Materials and Measures**

**ODD.** At T3, trained experimenters interviewed one of the child's parents using the Preschool Age Psychiatric Assessment (PAPA; Egger, Ascher, & Angold, 1999), a well-established semi-structured assessment of child psychopathology. This interview yielded both a dichotomous measure of ODD, together with a continuous variable indicating the cumulative presence of ODD symptoms. This measure has good reliability (Egger, Erkanli, Keeler, Potts, Walter, & Angold, 2006) and validity (Cicchetti, 2016). Inter-rater reliability figures for this measure were not available at the point of writing.

The presence or absence of each of the following eight symptoms of ODD was established for each child: often loses temper, often argues with adults, often actively defied or refuses to comply with adults' requests or rules, often deliberately annoys people, often blames others for his or her mistakes or misbehaviour, is often touchy or easily annoyed by others, is often angry and resentful, or is often spiteful or vindictive. The presence of four or more symptoms indicated a diagnosis of ODD.

**Maternal depression.** The Schedules for Clinical Assessment in Neuropsychiatry (SCAN; Wing et al., 1990) were used to determine diagnoses of Major Depressive Disorder (MDD) in mothers in pregnancy, before six-months postpartum, and in the past. Inter-rater

reliability between the trained interviewers and psychiatrists was evaluated with the kappa statistic, revealing good agreement regarding past ( $\kappa = .76, p < .001$ ), antenatal ( $\kappa = .78, p < .001$ ) and postpartum disorder ( $\kappa = .80, p < .001$ ).

**Maternal anger.** Three items measuring the frequency of overt anger expressions in the parental relationship were taken from the Iowa Family Interactions Rating Scale (Melby, Conger, Ge & Warner, 1995). Mothers were asked to rate the frequency with which they expressed anger in the context of their relationship, providing a self-report measure (*how often do you get angry at her/him? , how often do you shout at her/him because you were upset with her/him? and how often do you argue with your partner?*), whilst their partner provided a parallel measure of the mother's anger (*how often does your partner get angry at you? , how often does you partner shout at you because s/he was upset with you? and how often does your partner argue with you?*). A seven-point scale labelled as *always* through to *never* was provided. Previous analysis of a sample of this data confirms high test-retest reliability ( $r = .69, p < .001$ ), and good internal consistency ( $\alpha = .88$ ).

Mothers completed the Adult Wellbeing Scale (AWB; Snaith, Constantopolous, Jardine, & McGuffin, 1978) at T2. The AWB measures irritability, depression and anxiety. For the purposes of this analysis, three items pertaining to anger will be used: *I lose my temper and shout and snap at others, I feel I might lose control and hit or hurt someone, and people upset me so that I feel like slamming doors or banging about*. Mothers indicated whether each behaviour was true of them *definitely, sometimes, or never*. The internal consistency of these three items was questionable ( $\alpha = .63$ ), however alpha levels are difficult to interpret with small numbers of items.

Mothers and their infants were filmed in a two-minute mother-infant interaction task in the home at six months postpartum. Mothers were asked to interact with their infants using an activity board toy. This colourful plastic toy depicted a collection of animals and

corresponding textured fabrics, and opened in a book fashion. Time-sampling observation (every 5-seconds) of this interaction provided a measure of mothers' frustration in interaction with her child, providing a continuous measure of maternal frustration. Mother's vocal expression of anger was operationally defined as *impatience in the voice, lowered in pitch, a loud voice of harsh quality; or, speech under the breath*. The words spoken were not measured, but rather the tone with which they were spoken. A second observer coded a random 20% of the available cases (54/271), and an excellent level of inter-rater agreement was found according to the intra-class correlation statistic ( $ICC = .75$ ).

***Mothers' history of antisocial problems.*** A set of seven retrospective items measuring DSM-IV symptoms of oppositional defiant disorder and conduct disorder in childhood were incorporated into the pregnancy questionnaire. The presence of anger, fighting, disobedience, truancy, stealing, dishonesty, and vandalism was established. Mothers were also asked rate the presence of antisocial personality disorder symptoms in adulthood using the International Personality Disorder Examination screening instrument (IPDE; Loranger et al., 1994). For both their child and adult symptoms, the mother reported whether the behaviour was not true, somewhat true, or certainly true of them. Answers were summed across both questionnaires. The resulting composite variable showed a good level of internal consistency,  $\alpha = .79$ . This variable is hereto referred to as ASB.

***Social adversity.*** Measures of sociodemographic risk were collected at T1. These included; mother's age, educational attainments, social class, and marital status.

Dichotomous variables of 1 and 0 were created based upon each factor respectively: (1) entering parenthood as a teenager versus an adult, (2) comparing working and middle class families, (3) mothers without or with the basic qualifications expected in the UK, (4) unmarried versus married parents, and (5) stable versus unstable partnerships. To avoid problems of multicollinearity in the analyses, and in accordance with other publications from



the CCDS, a factor score based on the five dichotomous categories of social risk was used.

This scale showed an acceptable level of internal consistency,  $\alpha = .77$ .

### **Procedure**

Expectant mothers and their accompanying family members were approached by researchers in the hospital or clinics. Families were given a brief description of the nature of the CCDS and what their enrolment would entail. If the families expressed an interest they were asked to provide contact details so that an administrator could contact them one or two weeks later. During this follow-up call, the administrator arranged an appointment at home with those families who had decided to take part in the CCDS. This appointment was made for the third trimester of pregnancy (T1). T2 and T3 were also conducted in the family home, and involved questionnaires, interviews and observational tasks.

### **Analytic Strategy**

Descriptive statistics, zero-order correlations, and tests for outliers and non-normality were explored using SPSS (version 23). Subsequent analyses were conducted using Mplus (version 7.3). Structural equation modelling (SEM) was used to analyse the data with the aim to assess the fit of the hypothesised model. A benefit of SEM over other statistical techniques is its use of latent variables which account for measurement error, its ability to simultaneously model interrelationships between several variables, and to handle missing data.

Using a latent variable approach, the ‘maternal anger’ construct was estimated from the measured variables described above. Then, path models were planned to test a series of hypotheses regarding the longitudinal associations between the predictors and ODD, and to build a parsimonious model. As an adjunct to hypothesis testing, an exploratory approach to model building was taken when appropriate. Where hypotheses were not met, and the hypothesized models did not fit the data, alternative models were considered. This process

was both data- and theory-driven, and followed a pattern similar to that of a step-wise regression. Indices of fit were compared to establish the most accurate and parsimonious models in turn. The previously described hypotheses were tested.

- (a) Does antenatal depression predict ODD, accounting for prior depression and postnatal depression?
- (b) Is maternal anger directly related to child ODD?
- (c) Does maternal anger mediate the relationship between maternal depression and ODD?
- (d) Does the mothers' ASB account for any significant relationship above?
- (e) Is social adversity a predictor of ODD? Does social adversity moderate the relationship between maternal anger and ODD?
- (f) Is child gender a moderator in the prediction of ODD? Does child gender moderate the relationship between maternal anger and ODD?

Examination of indirect pathways was conducted according to the criteria set forth by McKinnon and colleagues (McKinnon, Krull, & Lockwood, 2000); mediation can be tested when the predictor and criterion variables are related to the proposed mediator. Consistent with Shrout and Bolger's (2002) recommendations, 1000 bootstrap samples were formed to estimate bias-corrected confidence intervals for the estimated indirect effects. Indirect effects were considered significant if the 95% confidence intervals did not include zero (Hayes, 2009; Preacher & Hayes, 2008). To test for the significance of child gender as a moderator, this dummy coded categorical variable was added in multiplication with the corresponding (mean-centred) predictor variable. A significant moderation was indicated when this interaction term was a significant predictor of the dependent variable. Social adversity was considered as a continuous moderator and was similarly multiplied with the predictor variables in turn, and the resulting interaction term added to the model to test for significance.

Where a significant moderation was found, the Johnson-Neyman (1936) technique was employed to determine the region of significance.

Where possible, indices of fit were assessed with: the chi-square ( $\chi^2$ ) statistic, root mean square error of approximation (RMSEA; .06 or less; Hu & Bentler, 1999), the standardized root mean square residual (SRMR; .08 or less; Hu & Bentler, 1999) and the comparative fit index (CFI; .95 or greater; Hu & Bentler, 1999) for the continuous outcome and categorical outcomes respectively. Changes in the values of the Akaike information criterion (AIC) Bayesian information criterion (BIC), and the sample-size adjusted BIC were also used to indicate whether model fit was improved. Where a categorical dependent variable was included, the robust weighted least squares approach was used (WLSMV). Models were run using delta parameterization as default, but theta parameterization was used where indicated. The results are presented according to guidelines for the reporting of SEM (Schreiber, Nora, Stage, Barlow, & King, 2006).

The final model derived from the analyses above was tested twice, once with the continuous symptom count, and again with the diagnostic ODD outcome. This approach has important implications for clinical services, which are designed to provide resources and support for individuals within a clinical, diagnostic realm. Indeed, DSM-V has continued to consider ODD as a categorical diagnosis. However, there remains considerable debate regarding whether ODD is dimensional or categorical in nature. Recent taxometric analyses examining the latent structure of ODD have found that ODD has a dimensional, rather than categorical, structure (Barry, Marcus, Barry, & Coccaro, 2013). As such, we analysed the data as dimensional, and tested the final model with the fully categorical diagnostic outcome criteria.

## Results

### Data Preparation

A total of 268/332 (80%) families remained in the study at T3. The 64 families lost to attrition did not differ from those in the final sample in terms of depression diagnosis, any maternal anger variable, or mothers' ASB. However, they showed significantly higher adversity scores,  $t = 2.834, p < .01$  ( $df = 90.16$ ). To test whether the 268 children differed from those who did not complete the study in relation to the ODD outcome, those with a completed PAPA versus those without were compared on their CBCL ODD factor score from age 3. The CBCL factor score was significantly associated with the ODD symptom count ( $r = .31, p < .001$ ) and ODD diagnosis  $t(225) = -3.96, p < .001$ . However, there was no significant difference in the CBCL score when comparing the children assessed and not assessed at age 7,  $t(252) = .27, p = .79$ . The approach for missing data estimation used depends upon whether the data are missing at random or the missingness is nonignorable. As the missingness of the outcome variable ODD is not related to the estimated values at an earlier time point, it is assumed that the data are missing at random (MAR). Therefore, in subsequent analyses, missing data were estimated using full information maximum likelihood estimation (FIML; Kline, 2005). This method is considered reasonable where data are at least missing at random (Arbuckle, 1996).

The data were inspected for multivariate normality using the Mahalanobis test ( $D^2$ ), a statistical measure that evaluates the position of each observation compared with the centre of all observations for the variables included in the proceeding analyses. Four observations exceeded the critical value of 39.252 at a p-level of .001 (max 51.87), indicating outliers. In addition, Mardia's test revealed that the data did not show a normal multivariate distribution. Therefore, a robust estimation method was used in order to account for non-normal data (Yuan & Bentler, 2000).

### Measurement Model

In order to develop the latent variable maternal anger, the 10 anger items were firstly assessed for zero-order correlations and descriptive statistics. Then, a series of confirmatory factor analyses were conducted, in order to establish the model that provided the best fit to the data, and the most accurate latent variable. Descriptive statistics are presented in Table 2, and the zero-order correlations for the indicator variables in Table 3.

Table 2.

*Descriptive Statistics for Variables Included in the Confirmatory Factor Analyses.*

Variable	Range	Actual range	<i>M</i>	<i>SD</i>
1. Lose temper	0 - 3	0 - 3	1.11	0.79
2. Out of control	0 - 3	0 - 3	0.17	0.46
3. Feel rage	0 - 3	0 - 3	0.58	0.70
4. Anger	1 - 7	1 - 7	3.20	1.21
5. Shouts	1 - 7	1 - 7	2.85	1.35
6. Argues	1 - 7	1 - 7	3.66	1.46
7. Anger (partner rated)	1 - 7	1 - 7	3.27	1.39
8. Shouts (partner rated)	1 - 7	1 - 7	2.93	1.47
9. Argues (partner rated)	1 - 7	1 - 7	3.49	1.50
10. Observed frustration	0 - 100	0 - 33.33	2.17	4.71

Table 3.

*Zero-Order Correlations and Factor Loadings for Latent Variable Indicators.*

Variable	2	3	4	5	6	7	8	9	10	Factor Loadings	
										10-item	8-item
1. Lose temper	.31***	.48***	.48***	.50***	.49***	.32***	.33***	.34***	.17*	.55	.53
2. Out of control	-	.32***	.28***	.26***	.22**	.15	.17*	.16	.07	.34	-
3. Feel rage		-	.32***	.38***	.38***	.19*	.22**	.22**	.04	.44	.40
4. Anger			-	.75***	.61***	.57***	.54***	.51***	.21*	.82	.90
5. Shouts				-	.60***	.50***	.53***	.50***	.23**	.81	.90
6. Argues					-	.34***	.38***	.47***	.12	.66	.67
7. Anger (partner rated)						-	.79***	.72***	.23**	.79	.60
8. Shouts (partner rated)							-	.72***	.23**	.79	.59
9. Argues (partner rated)								-	.17*	.78	.57
10. Observed frustration									-	.26	-

Note. \* =  $p < .05$ , \*\* =  $p < .01$ , \*\*\* =  $p < .001$

The hypothesised 10-item factor incorporating all measures of maternal anger (CFA model 1, Table 4) did not provide a good fit to the data. The zero-order correlations suggested that observed frustration showed low associations with all other anger variables, as did the 'out of control' item. Furthermore, the individual factor loadings suggested that the items of observed frustration and loss of control were not reliably related to the overall factor; they both demonstrated a factor loading below the cut-off of .4 (Stevens, 1992). Therefore, these were removed sequentially (CFA models 2 and 3 in Table 4), and the fit compared. Removing observed frustration and loss of control did not significantly improve model fit according to the chi-square values. However, the AIC and BIC values decreased. In addition, the Cronbach's alpha level suggested the internal consistency was much improved. Modification indices indicated correlated measurement errors between the father-rated items, mother's shouting and anger, and her self-rated rage and temper respectively. These items were from the same respective questionnaires; therefore, their correlated errors may have been due to the similar vocabulary used between the items. By allowing the error terms to be correlated at this step, the model fit was improved (model 4 in Table 4). The final combination of 8 indicator variables (Figure 2) reflects a good fit of the model to the data, and shows good internal consistency (Cronbach's  $\alpha = .87$ ; George and Mallery, 2003). The final maternal anger variable showed a significant positive association with the separate observed frustration item,  $r(320) = .34, p < .01$ .

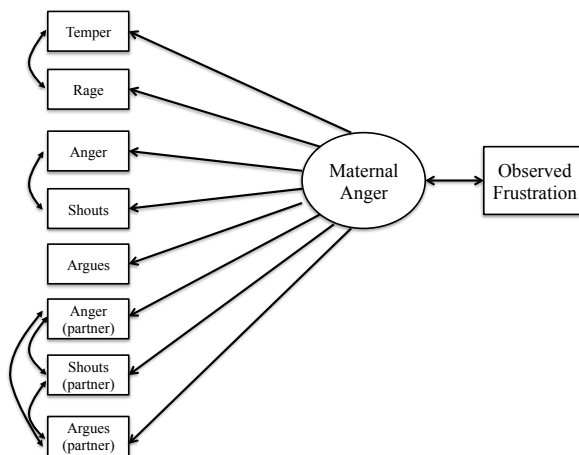


Figure 2. Final measurement model of maternal anger and observed maternal frustration.

Table 4.

*Fit Indices for the CFA Models for Maternal Anger.*

Model	CFI	TLI	SRMR	RMSEA	95% CI for RMSEA	$\chi^2$	df	<i>p</i>	AIC	BIC	Sample size adj BIC	Models compared	$\Delta\chi^2$	<i>p</i>
1	0.763	0.696	0.085	0.133	0.116, 0.150	219.704	35	<.001	6952.21	7063.33	6968.19			
2	0.748	0.664	0.093	0.161	0.141, 0.181	219.073	27	<.001	5313.35	5411.00	5325.39	1,2	-0.631	ns
3	0.744	0.641	0.094	0.182	0.159, 0.205	201.566	20	<.001	5014.55	5101.35	5025.26	1,3	-17.507	ns
4	0.983	0.969	0.028	0.054	0.017, 0.086	26.841	15	<.05	4848.99	4953.88	4861.92	1,4	-174.725	<.001

Descriptive statistics and zero-order analyses of the variables considered in the subsequent models are presented in tables 5 and 6. The CCDS sample is nationally representative with respect to sociodemographic characteristics of first time parents. In addition, rates of antenatal and postnatal depression were in line with the UK population norms. Rates of ODD were similarly representative. Therefore, the sample population is generalisable to the UK as a whole.

Table 5.

*The Means, Standard Deviations and Ranges for Additional Variables Included in the Structural Models.*

Variable	N	Range	Actual range	<i>M</i>	<i>SD</i>
Adversity	332	1.0, 2.50		0	1
Antisocial history	332	0, 20	0, 20	4.44	4.09
DSM ODD symptoms	268	0, 8	0, 7	2.18	4.71
Child ODD (n, %)					
ODD	31 (11.6%)				
No ODD	237 (88.4%)				
Maternal depression (n, %)					
Depressed in past	84 (25%)				
Not depressed in past	248 (75%)				
Depressed in pregnancy	54 (16%)				
Not depressed in pregnancy	278 (84%)				
Depressed postpartum	34 (11%)				
Not depressed postpartum	272 (89%)				
Child gender (n, %)					
Female	142 (43%)				
Male	186 (57%)				



Table 6.

*The Zero-Order Associations Between Variables Included in the Structural Models.*

	1	2	3	4	5	6	7	8	9	10
1. ODD symptoms	-	.79**	.12*	.06	.05	.04	-.01	.15**	.24***	.20**
2. ODD diagnosis		-				.07	.01	.18**	.19**	
3. Prior depression		.26	-	.19***	.11 †	.03	.15*	.05	.10 †	
4. Antenatal depression		2.25		-	.35***	.14 †	.21**	.44***	.36***	
5. Postnatal depression		.14			-	.06	-.01	.24***	.31***	
6. Maternal anger (latent)						-	.11	.32***	.28***	.03
7. Observed frustration							-	.32***	.16**	-.04
8. Adversity								-	.49***	.10
9. Antisocial history									-	.11
10. Child gender		6.74*	.06	.24	.48					-

Note. \*= $p < .05$ , \*\*= $p < .01$ , \*\*\*= $p < .001$ , †= $p < .10$

Numbers above the diagonal are correlations (point-biserial when the dichotomous variables 2,3,4,5, and 10 are correlated with other variables). Numbers below the diagonal (where the association between two dichotomous variables is tested) are the Pearson's chi-square test statistic, with significance calculated using the Fisher-exact test. Dummy coded variables are gender (male = 1, female = 0), ODD diagnosis (ODD = 1, no ODD = 0), and all depression variables (depression = 1, no depression = 0).

## Hypothesis Testing

A series of models were tested, each based on theory and empirical findings from the analyses. First, a simple model testing the direct pathways between antenatal depression, postnatal depression, maternal anger, observed frustration, and ODD symptoms was estimated (model 1). The path coefficients for this model are shown in Figure 3, and the indices of fit in Table 7. This model showed a good fit. Mothers' antenatal depression was positively associated with their expression of anger postpartum, and with experimenter-rated frustration in the postpartum period. Depression in the postpartum period was not related to the contemporaneously measured maternal anger, nor to any other variable of interest. Therefore, it was removed from subsequent models. The revised model (Figure 3; model 2) demonstrated better model fit according to the reduced AIC and BIC values (see table 7). However, although antenatal depression was prospectively related both to maternal anger and observed frustration, antenatal depression, maternal anger and frustration were not related to

ODD symptoms at age 7. Thus, the prerequisites for the hypothesized indirect effects were not met (the proposed mediators, maternal anger and observed frustration, were not associated with the outcome variable, ODD symptoms). Therefore, an exploratory approach to model building was taken that was both empirically and theoretically informed, rather than the procedure set forth in Figure 1.

### **Predicting ODD: Exploratory Model Building**

Zero-order correlations demonstrated that mothers' prior depression was significantly associated with child ODD symptoms. Evidence of a past depressive episode may indicate a chronicity or recurrence of depression, therefore the impact of past depression was estimated by adding this to the model. A significant direct association between prior depression and ODD symptoms was found, but this did not occur through its association with antenatal depression. Therefore, the path between antenatal depression and ODD was constrained (Figure 3; model 3). This resulted in a good model fit, but it did not improve upon the previous model (Table 7). Prior depression was not related to mothers' anger postnatally, but showed a significant, unexpected negative relationship with observed frustration; mothers who had been depressed in the past demonstrated less frustration than those who had not been depressed<sup>1</sup>. A significant relationship in the opposing direction was true for antenatal depression; mothers who were depressed antenatally had greater maternal anger and observed frustration postnatally.

Next, the significance of the indirect effects between past depression, antenatal depression and (1) maternal anger and (2) observed frustration were tested. There was a significant indirect association between past depression and maternal anger via antenatal depression ( $\beta = .05, p < .01$ ), and the confidence intervals confirmed that the relationship was

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<sup>1</sup> The association between prior depression and observed frustration was also negative at the univariate level, therefore there did not appear to be a suppressor effect.

significant, 95% CI [.01, .09]. The significant Sobel test statistic further supports this mediation ( $z = 2.41, p < .05$ ), and the nonsignificant direct relationship suggests that antenatal depression fully mediated this pathway ( $\beta = .01, p = .92$ ); past depression was related to maternal anger through antenatal depression. There was also a significant indirect relationship between past depression and observed frustration via antenatal depression ( $\beta = .05, p < .01$ ), confirmed by the confidence intervals, 95% CI [.01, .09]. The Sobel statistic further supported this mediation ( $z = 2.65, p < .01$ ). However, the direct relationship between past depression and observed frustration remained significant when antenatal depression was accounted for,  $\beta = -.14$  (95% CI  $-.22, -.07$ ), suggesting antenatal depression only partially mediated the relationship between past depression and observed frustration.

**Mothers' ASB.** Next, because mother's ASB showed the expected positive relationship with child ODD symptoms and to maternal depression in univariate analyses, it was added to the model (Figure 3; model 4). This model demonstrated good fit (Table 7). Mother's ASB had a significant direct effect on child ODD symptoms ( $\beta = .26, p < .001$ ) and was prospectively related to maternal anger, but not observed frustration. Although ASB was related to past depression, mothers' past depression no longer demonstrated any association with child ODD.

Because past depression no longer explained relationships with maternal anger, observed frustration, or child ODD, past depression was removed to create a more parsimonious and statistically sound model (model 5), and the chi-square statistic decreased (Table 7). Mother's ASB had a direct positive association with maternal anger ( $\beta = .39, p < .001$ ) but still not with observed frustration ( $\beta = .11, p = .09$ ). Antenatal depression partially mediated the relationship between mothers' ASB and postpartum anger ( $z = 2.14, p < .05$ ); there was a significant indirect effect ( $\beta = .06, p < .05$ ), 95% CI [.001, .12], but the significant direct effect remained ( $\beta = .33, p < .05$ ). Despite the nonsignificant direct

relationship between maternal ASB and mothers' frustration, there was a significant indirect pathway via antenatal depression ( $\beta = .06, p < .05$ ), 95% CI [.003, .12]. A Sobel test confirmed the presence of mediation ( $z = 2.33, p < .05$ ).

**Social adversity.** Next, social adversity was considered as a continuous moderator between the two mediating variables (maternal anger and observed frustration) and child ODD respectively, as well as a direct predictor of ODD. Social adversity moderated the relationship between maternal anger and child ODD (model 6), but did not moderate the relationship between observed frustration and ODD. The Johnson-Neyman technique (Johnson & Neyman, 1936) was used to establish the region of significance for the simple slope of maternal anger on ODD symptoms, adjusted for the values of the continuous moderator of adversity. This revealed that there was a significant relationship between maternal anger and child ODD symptoms, but only when adversity was high (above the mean, representing 40.2% of the sample). For these families, maternal anger was negatively associated with child ODD; less maternal anger was required to predict child ODD symptoms as adversity increased. When adversity was lower than 0.5 standard deviations above the mean, maternal anger had no significant association with ODD symptoms. Appendix C illustrates the conditional direct effect ( $\beta$  coefficient) at all values of the moderator with a 95% confidence interval band.

**Child gender.** Next, child gender was considered as a potential moderator in the relationship between maternal anger/observed frustration and child ODD, and as a direct predictor of ODD. Of the 31 children who met the criteria for oppositional defiant disorder, the majority of those children were boys ( $n = 24/31, 77.4\%$ ). There was a significant association between gender and ODD in terms of diagnosis ( $\chi^2 = 6.72, p < .01$ ) and symptom count ( $t = -3.40, p < .001$ ). Girls had fewer ODD symptoms on average,  $M = 0.90$  ( $SD = 1.31$ ) than boys,  $M = 1.57$  ( $SD = 1.72$ ). Accordingly, when added to the model (model 7) child

gender showed a direct effect on ODD symptoms; male infants were more likely to display greater ODD symptom levels, therefore this was added to the model. There was no notable change in the relevance of other variables in predicting ODD after gender was added. There was no significant moderation of child gender in the associations between either maternal anger or observed frustration with child ODD (the interaction terms were not significant in each case).

Observed frustration was not found to be associated with ODD symptoms either directly or in interaction with either child gender or social adversity. Removing observed frustration from the model resulted in an improved model fit according to the AIC and BIC (model 8).

**ODD as a diagnostic categorical outcome.** Finally, the resulting model was tested using ODD diagnosis, rather than symptom level, as the primary outcome variable. As the outcome variable of ODD diagnosis was dichotomous, the weighted least squares estimator was used; parameter estimates and their standard errors would be too low with maximum likelihood estimation (DiStefano, 2002). The resulting model demonstrated a good overall fit (Figure 5; model 9). Furthermore, the moderated relationship between maternal anger and ODD as a function of social adversity remained significant, as did the direct relationship between maternal ASB and child ODD.

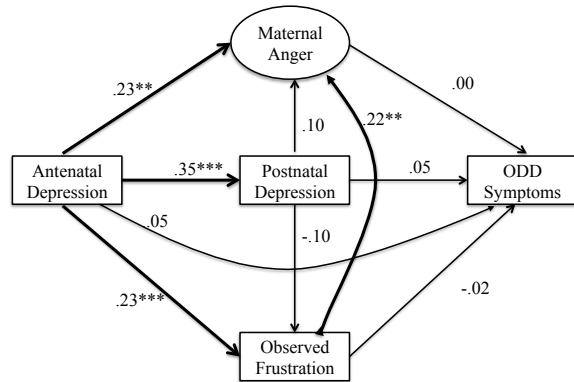
Table 7.

*Model Fit Indices for each Structural Model.*

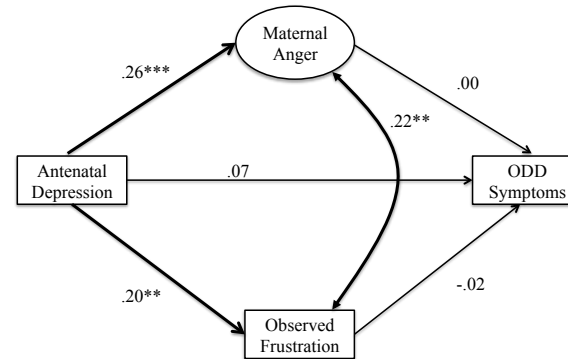
Model	CFI	TLI	SRMR	RMSEA	95% CI for RMSEA	$\chi^2$	df	<i>p</i>	AIC	BIC	Sample size adj BIC
1	0.975	0.961	0.049	0.039	0.016, 0.058	63.86	43	<.05	7617.16	7786.60	7643.87
2	0.980	0.973	0.044	0.035	0.000, 0.057	50.08	36	ns	7488.03	7634.25	7510.56
3	0.985	0.978	0.042	0.029	0.000, 0.049	56.02	44	ns	7751.90	7919.32	7779.76
4	0.981	0.971	n/a	0.024	0.000, 0.045	61.04	51	ns			
5	0.976	0.964	n/a	0.029	0.000, 0.050	56.38	44	ns			
6									7710.06	7881.29	7738.55
7									7692.95	7867.43	7721.52
8									6055.91	6207.63	6080.75
9									5259.82	5407.75	5284.04

*Note.* Various fit indices are provided according to the estimation method used. In models 1-3, the MLR approach was taken, and all fit indices were available. For models 4 and 5, the WLSMV approach was taken, for which the SRMR was not available. This approach was continued for the remaining models, yet fit indices were not available when an interaction with a latent variable was estimated; these fit statistics are only available whenever means, variances and covariances are sufficient statistics for model estimation. In these, the AIC and BIC are provided.

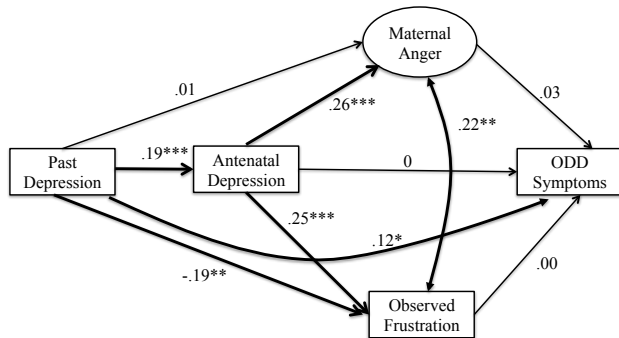
Model 1



Model 2



Model 3



Model 4

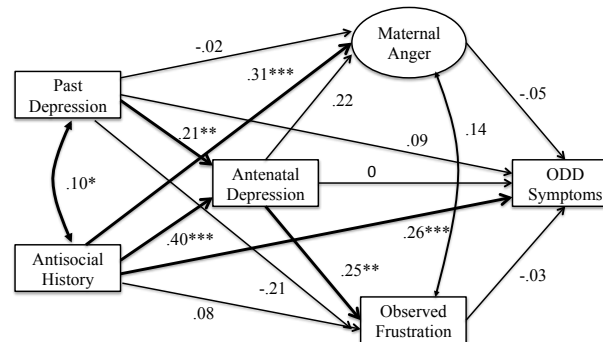
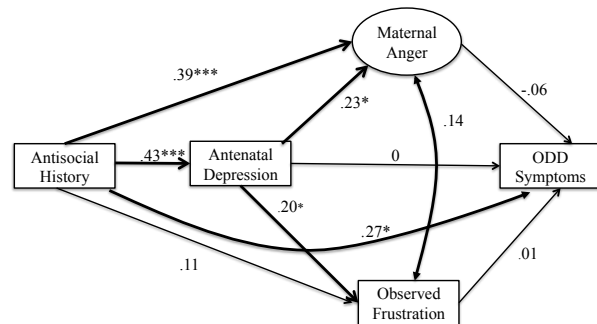
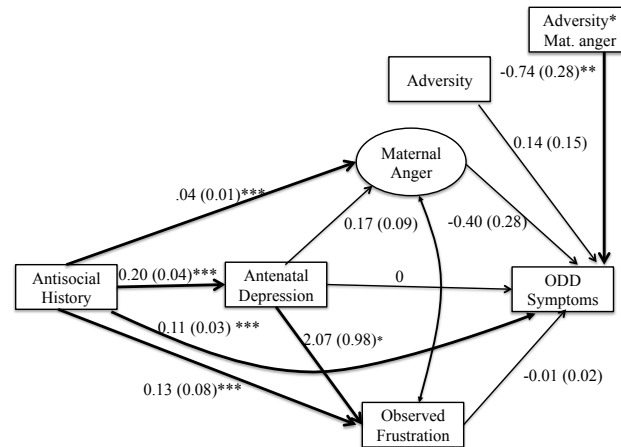


Figure 3. Models 1 through 4.

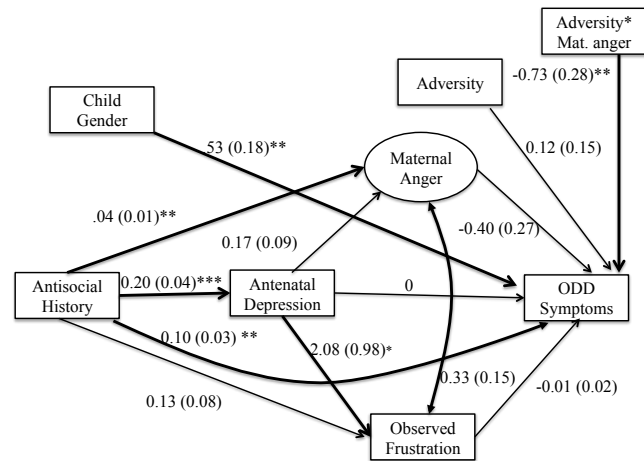
Model 5



Model 6



Model 7



Model 8

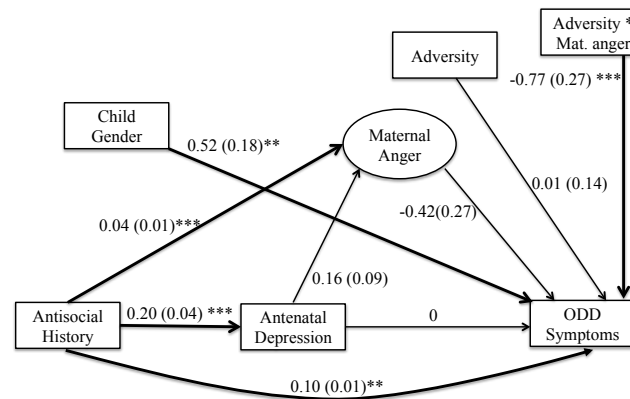


Figure 4. Models 5 through 8 (unstandardized coefficients and SE are presented in models 6, 7 and 8).



Model 9

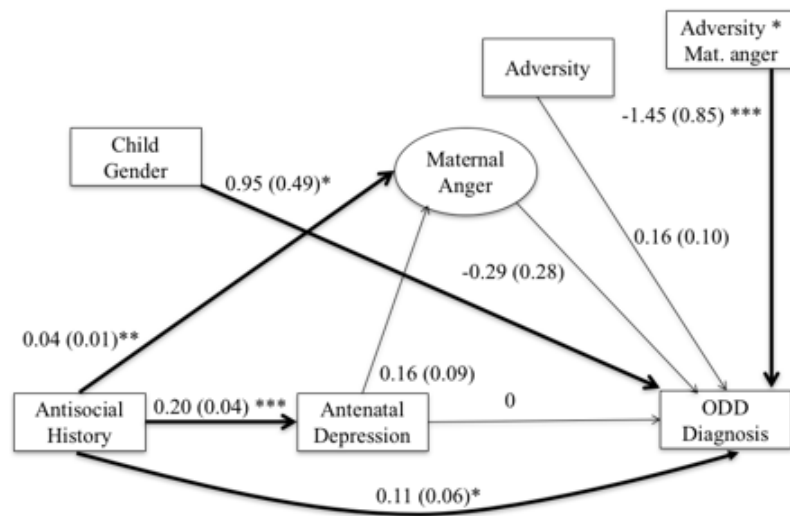


Figure 5. Model 9.

## Discussion

The main hypothesis, that maternal anger would mediate the relationship between antenatal depression and child ODD, was not initially supported; mothers with and without antenatal depression had children who did not differ in the number of ODD symptoms. In addition, maternal postpartum depression showed no association with child ODD. Mother's history of previous depressive episodes was associated with ODD symptoms; children of mothers with a history of at least one episode of depression prior to their first pregnancy showed a greater number of ODD symptoms. However, this relationship was no longer significant once the mothers' ASB was accounted for; indeed, an antisocial history was related to maternal depression across the perinatal period. This finding differs from previous studies which present a significant association between maternal depression and externalizing difficulties (e.g. Hay, Pawlby, Waters, Perra, & Sharp, 2010; Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005; O'Donnell, Glover, Barker, & O'Connor, 2014; Pemberton et al.,

2010), and highlights the importance of considering other factors that may play a part in the prediction of, in this case, ODD.

The proposed latent anger variable, along with the unproposed separate variable of observed frustration, did not show the hypothesised positive relationship with child ODD, and therefore neither were considered to be mediators between antenatal depression and ODD. However, maternal anger was differentially related to child ODD as a function of social adversity. In families with low levels of adversity, maternal anger was not significantly related to ODD. However, when adversity was higher, maternal anger showed a significant relationship with ODD; as adversity increased, lower levels of maternal anger were required to predict ODD. This suggests that children are more sensitive to the impact of maternal anger in social environments of lower maternal education and age. Given that the majority of studies previously described have recruited high- risk samples, this representative sample-based finding adds to the existent literature by demonstrating that the relationships between risk factors (in this case, maternal anger) and externalizing outcomes differ according to social risk.

Maternal ASB, that is their self-reported levels of externalizing symptoms in childhood and adulthood, emerged as an important independent predictor of child ODD. That is, mothers with a higher frequency of childhood and adulthood externalizing symptoms had children with greater levels of ODD symptoms by age 7, or indeed children who met the diagnostic threshold. In accordance with previous literature, these mothers were also more likely to experience depression in the antenatal period, as well as prior to their pregnancy with their firstborn. Mothers with greater ASB also showed higher levels of maternal anger. Indeed, an unfolding pattern emerged in the model, whereby mother's past antisocial history predicted their antenatal depression, which then predicted maternal anger postpartum (although the direct effect also remained), and is in accordance with previous literature. The

same unfolding pathway between antisocial history and observed frustration via antenatal depression was demonstrated.

Contrary to previous literature, there was no difference in the relationship between maternal anger or frustration and ODD according to the gender of the child. However, as the number of female infants with ODD was significantly less than of boys, the small cell size may have limited the power to detect any effects. In accordance with the majority of the previous literature, there was a direct prediction of ODD by child gender.

The nonsignificant relationship between maternal depression and child ODD, particularly when measured in the perinatal period, was somewhat unexpected given the findings in previous literature. A number of explanations for this may be worthy of consideration. Firstly, previous studies have measured both maternal depression and child ODD using the same informant. Such a method may inaccurately inflate the size of the effects. While mothers were interviewed for their depression scores, and also provided the data regarding their children's ODD symptoms, an independent interviewer and rater assessed each of these, therefore reducing the bias that may have inflated the findings of previous literature that had employed self-report measures (Waters, Hay, Simmonds, & van Goozen, 2014). Secondly, ODD symptoms are distinct from normative childhood behaviours by their frequency and perseverance, and yet many previous studies documenting the associations between antenatal depression and antisocial outcomes have used dimensional measures that include a wide range of behaviour severity that may include normal behaviours. Whilst a worthy endeavor, the data here, based on diagnostic measurement, suggest that paying attention to the differential patterns in predicting normative and diagnostic levels of ODD symptoms is an important distinction to make; the risk factors that predict more normative differences in behavior may be different to those that predict diagnostic outcomes. Thirdly, Interestingly, associations between maternal depression and

more severe diagnostic outcomes, such as conduct disorder and adult/teenage violence, have been shown relatively consistently. Therefore, there may be differential effects of maternal depression depending on the severity and type of externalizing outcome (from ODD to CD) that are not linear in nature.

The families who have participated in the CCDS represent the UK population in terms of demographics, and in terms of depression and ODD diagnostic prevalence. This offers a unique opportunity to consider the interplay between various factors across a sample that includes both low and high-risk families. Importantly, it means that the results are more likely to be generalisable to the population within which the participants are drawn; there is no bias that may be inflating the relationships between factors of interest. However, as a result, it is possible that a larger sample size may be a requirement to increase the power to detect a true effect in a representative sample. The sample in the current study was sufficiently powered to detect the small effect demonstrated in previous studies that have examined the association between maternal depression and child externalizing problems in higher risk samples (for example,  $r = .21$ ; Goodman et al., 2011). Indeed, a priori analyses suggested a sample of 62 was sufficient to detect this small effect. The cost of conducting a larger study that would retain the level of quality of measurement (for example, using standardized interviews to determine diagnoses) would be significant. However, any null effects in the current study should be considered within the context of the statistical limitations

Where significant effects have been found, the strength of the relationships between the predictive variables (interpretable where standardized coefficients are presented) is low, and that is somewhat expected. This suggests there are likely to be other explanatory factors in predicting ODD that are not included in the current model. For example, the potential influence of paternal depression, anger, and antisocial history is neglected here. Mothers with

an antisocial history are more likely to have relationships with antisocial men (Krueger, Moffitt, Caspi, Bleske, & Silva, 1998), therefore this may be an unmeasured third variable. Maternal depression may influence ODD through its chronicity or severity; more detail regarding the chronicity of depression may reveal that subsequent or continued episodes of maternal depression may impact on the mother across the child's life, and indeed upon their experience of anger over time, beyond the six-months postpartum as measured in this study.

The observed measure of frustration showed good reliability between raters, but was not a measure consistent with the maternal anger latent variable. This may be for many reasons. The situational and transient nature of the observation may have led to increased measurement error in comparison to the more stable questionnaire measure; mothers' expression will likely have been affected by factors unrelated to their trait levels of frustration (such as tiredness), thereby reducing the accuracy of the measure. Despite its differentiation from the maternal anger variable, observed frustration was significantly correlated with maternal anger. This suggests that they are not orthogonal constructs, and offers some validation to the self- (and partner-) reported measure of maternal anger (and vice versa). However, it may be that frustration and anger are distinct enough to require independent study. Both constructs were also similarly related to antenatal depression; mothers who were depressed during their pregnancy were more likely to report higher anger and to demonstrate higher frustration in interaction with their infant at six-months postpartum. Of note, the concurrent depression measure did not explain this association, and offers further evidence for the importance of the antenatal period in shaping the postnatal experience for women.

### **Summary**

In sum, this study provides a description of the potential interplay between maternal depression, maternal anger, and mothers' ASB history in the prediction of ODD in their

children. Whilst some expected relationships were not supported, the representative nature of the sample makes it distinct from many other previous studies of the antecedents of ODD.

The findings imply there is an association between maternal depression and anger, and that this is driven by mothers' antisocial history. This data adds support, therefore, for interplay between internalizing and externalizing disorders. The data is also consistent with previous literature that posits that the antenatal period is an important one for the development of parenting; maternal anger and frustration postpartum are associated with antenatal depression, and not postpartum depression. Therefore, future studies that consider the antecedents of psychopathology in children should consider the antenatal period and the parents' previous history of difficulties, in best explaining outcomes.

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## **Appendices**

Appendix A. Manuscript preparation and submission for the JCPP.

Appendix B. Dissemination statement.

Appendix C. Johnson-Nymen moderation.



## Appendix A

### Manuscript preparation and submission for the JCPP.

Papers should be submitted online. For detailed instructions please go to: <http://mc.manuscriptcentral.com/jcpp-camh>. Previous users can check for existing account. New users should create a new account. Help with submitting online can be obtained from the Editorial Office at [JCPP@acamh.org](mailto:JCPP@acamh.org)

1. The manuscript should be double spaced throughout, including references and tables. Pages should be numbered consecutively. The preferred file formats are MS Word or WordPerfect, and should be PC compatible. If using other packages the file should be saved as Rich Text Format or Text only.
2. Papers should be concise and written in English in a readily understandable style. Care should be taken to avoid racist or sexist language, and statistical presentation should be clear and unambiguous. The Journal follows the style recommendations given in the *Publication manual of the American Psychological Association* (5th edn., 2001).
3. The *Journal* is not able to offer a translation service, but, authors for whom English is a second language may choose to have their manuscript professionally edited before submission to improve the English. A list of independent suppliers of editing services can be found at [http://authorservices.wiley.com/bauthor/english\\_language.asp](http://authorservices.wiley.com/bauthor/english_language.asp). All services are paid for and arranged by the author, and use of one of these services does

not guarantee acceptance or preference for publication.

*Layout Title:* The first page of the manuscript should give the title, name(s) and short address(es) of author(s), and an abbreviated title (for use as a running head) of up to 80 characters.

*Abstract:* The abstract should not exceed 300 words and should be structured in the following way with bold marked headings: Background; Methods; Results; Conclusions; Keywords; Abbreviations. The abbreviations will apply where authors are using acronyms for tests or abbreviations not in common usage.

*Key points:* All papers should include a text box at the end of the manuscript outlining the four to five Key (bullet) points of the paper. These should briefly (80-120 words) outline what's known, what's new, and what's clinically relevant.

*Headings:* Articles and research reports should be set out in the conventional format: Methods, Results, Discussion and Conclusion. Descriptions of techniques and methods should only be given in detail when they are unfamiliar. There should be no more than three (clearly marked) levels of subheadings used in the text.

*Acknowledgements:* These should appear at the end of the main text, before the References.

*Correspondence to:* Full name, address, phone, fax and email details of the corresponding author should appear at the end of the main text, before the References.

*References* The *JCPP* follows the text referencing style and reference list style detailed in the *Publication manual of the American Psychological Association* (5th edn.)<sup>i</sup>.

*References in text:* References in running text should be quoted as follows:

Smith and Brown (1990), or (Smith, 1990), or (Smith, 1980, 1981a, b), or (Smith & Brown, 1982), or (Brown & Green, 1983; Smith, 1982).

For up to five authors, all surnames should be cited in the first instance, with subsequent occurrences cited as et al., e.g. Smith et al. (1981) or (Smith et al., 1981). For six or more authors, cite only the surname of the first author followed by et al. However, all authors should be listed in the Reference List. Join the names in a multiple author citation in running text by the word 'and'. In parenthetical material, in tables, and in the References List, join the names by an ampersand (&). References to unpublished material should be avoided.

*Reference list:* Full references should be given at the end of the article in alphabetical order, and not in footnotes. Double spacing must be used.

References to journals should include the authors' surnames and initials, the year of publication, the full title of the paper, the full name of the journal, the volume number, and inclusive page numbers. Titles of journals must not be abbreviated and should be italicised.

References to books should include the authors' surnames and initials, the year of publication, the full title of the book, the place of publication, and the publisher's name.

References to articles, chapters and symposia contributions should be cited as per the examples below:

Kiernan, C. (1981). Sign language in autistic children. *Journal of Child Psychology and Psychiatry*, 22, 215-220.

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Use Ed.(s) for Editor(s); edn. for edition; p.(pp.) for page(s); Vol. 2 for Volume 2.

### *Tables and Figures*

All Tables and Figures should appear at the end of main text and references, but have their intended position clearly indicated in the manuscript. They should be constructed so as to be intelligible without reference to the text. Any lettering or line work should be able to sustain reduction to the final size of reproduction. Tints and complex shading should be avoided and colour should not be used unless essential. Figures should be originated in a drawing package and saved as TIFF, EPS, or PDF files. Further information about supplying electronic artwork can be found in the Wiley-Blackwell electronic artwork guidelines at [http://authorservices.wiley.com/prep\\_illust.asp](http://authorservices.wiley.com/prep_illust.asp)

### *Nomenclature and symbols*

Each paper should be consistent within itself as to nomenclature, symbols and units. When referring to drugs, give generic names, not trade names. Greek characters should be clearly indicated.

**Appendix B****Dissemination Statement****1. Journal Publication**

It is expected that the study will be submitted for publication with the Journal of Child Psychology and Psychiatry.

**2. Presentation**

In June 2016, I will present my research findings to an academic audience as part of the Doctorate in Clinical Psychology at the University of Exeter.

**Appendix C****Johnson-Neyman Continuous Moderation**